different methods of induction, AF induced by Bu and A show similar moderate consistency of short-term activation direction which differs markedly from VF. 3) Geometric or mechanistic differences may be responsible for these observations.

1072-170 The Use of Bio-Battery Cell Output to Predict Lesion Formation and Prevent Rapid Impedance Rise

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When the electrodes of two metals are placed in contact with tissue, an electric current is produced that is directly proportional to the temperature at the tissue-electrode interface. Using this technique, we have previously shown that it is feasible to monitor temperature at the electrode-tissue interface without the use of thermistors or thermocouples. Bio-battery cell current reaches a maximum level followed by a decrease before the steep rise in impedance is seen. This experiment was performed to observe the temperature at which this occurs and to evaluate whether maximum cell output predicts lesion formation.

Methods: A 7-F EPT catheter with a thermistor mounted on the tip of a 4 mm distal electrode was used. Fresh bovine ventricular myocardium was submerged in a temperature controlled bath with circulating bovine blood. In the first protocol, radiofrequency (RF) energy was applied at a constant level of 30V. RF application was terminated when cell output decreased below 20% of peak bio-battery level. In the second protocol, RF energy was raised in a step wise fashion until there was a steep rise in impedance. Lesions were measured grossly and were stained with nitro blue tetrazolium. A total of 12 energy applications were made.

Results: The temperature at maximum bio-battery cell output was 70.8 ± 3.8°C with both protocols and there was a linear correlation between the cell voltage and the thermistor temperature ($r = 0.96 \pm 0.02$). Maximum cell output, followed by a 20% drop, resulted in lesion formation at all the ablation sites with mean lesion dimensions of 6.5 × 5.4 × 5.4 mm (length × width × depth) and there was no difference in lesion size in these two protocols.

Conclusion: Maximum bio-battery cell output predicts lesion formation. Maximum cell voltage may be used to provide a feedback loop to decrease the RF energy level to prevent tissue charring and rapid impedance rise.

1072-171 The Appropriate Isthmus Size for Maze Procedure

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Background: The goal of maze procedure is to prevent atrial fibrillation but allow sinus impulses to propagate via an isthmus, which connects viable atrial tissues. The size and structure of isthmus associated with this desirable effect remain unclear.

Methods: Endocardial isthmuses of decreasing sizes (2, 1 and 0.5 cm) were created in five isolated and perfused canine right atria (3.8 by 3.2 cm) in the presence of 1–5 uM of ACh. The cuts spared the epicardium. The tissues were paced at increasing rates from either site of the isthmus, and the resultant activation pattern was mapped from the epicardium using 477 bipolar electrodes (1.6 mm apart).

Results: With isthmuses 2 & 1 cm wide, regular pacing at CL of 150 ms from either side of the cut allowed impulses to conduct to the other side across the isthmus. Rapid pacing could induce either multiple WFs with "fibrillation"-type activity, or single stationary RWF (CL: 106 ± 17 ms) with "flutter"-type activity. Both types of WFs were able to propagate across the isthmus. With an isthmus of 0.5 cm, only single stationary RWF with a longer CL (138 ± 23 ms, $p < 0.05$) could be induced. This RWF consistently failed to propagate across the isthmus, while paced WFs could conduct in 1:1 fashion down to CL of 140–160 ms (n = 3) with pectinate muscle (PM) in the isthmus, or to CL of 250–300 ms (n = 2) without PM in the isthmus.

Conclusions: An endocardial isthmus of 0.5 cm prevents the propagation of RWFs and the generation of multiple WFs, while allowing paced WFs to conduct. The presence of PM in the isthmus increases the safety factor for impulse propagation at regular pacing.

1072-172 Dispersion of Atrial Refractoriness and Atrial Fibrillation Vulnerability: Relationship to Anatomic Site and Basic Cycle Length

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Background: An animal model of right atrial pacing in order to study atrial fibrillation (AF) is widely used, but the mechanisms of the development of a reversible atrial myopathy are poorly understood. We hypothesize that a different degree of dispersion of refractoriness and vulnerability for induction of non-sustained AF between trabeculated (T) and smooth right atrium (SRA) may contribute to initiation and maintenance of AF.

Methods: In 12 healthy closed-chest mongrel dogs weighing 22.4 ± 1 kg, programmed stimulation was performed from 2 sites on the TRA and 2 on the SRA. A premature beat was introduced beginning in late diastole, in 10 ms steps until effective refractoriness (ERP) was reached, using a basic cycle length (CL) of 400 and 300 ms and a current strength of 1, 3, 6 and 10 mA. AF vulnerability duration (VUL) was defined as the interval between first and last induction of AF (>5 repetitive atrial impulses, CL < 120 ms).

Results: There were no site-related differences in pacing threshold between TRA and SRA (0.29 ± 0.25 vs 0.33 ± 0.24 mA).

Pace site/CL (ms)	TRA/400	SRA/400	TRA/300	SRA/300
ERP (ms)	97 ± 17	100 ± 25	96 ± 14	98 ± 22
SD ERP (ms)	17 ± 5[*]	25 ± 6[*]	14 ± 5[†]	22 ± 7[†]
AF VUL (ms)	32 ± 22[‡§]	42 ± 29[‡]	23 ± 21[‡§]	35 ± 25[‡]

SD = standard deviation. [*], [†] = $p < 0.05$, [‡] = $p < 0.01$.

Conclusion: The dispersion of refractoriness is wider, and the vulnerability for induction of AF is increased on the smooth, as compared to the trabeculated RA, especially during slow pacing rates. These findings may contribute to initiation and maintenance of AF in the canine model of rapid RA pacing.

1073 Ventricular Tachycardia: Drugs; Trials

Monday, March 30, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 4:00 p.m.–5:00 p.m.

1073-173 Clinical Predictors of Antiarrhythmic Drug Response in the MUSTT Trial

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Background: Patients (pts) with drug refractory ventricular tachycardia (VT) are at high risk for sustained arrhythmias and death. However, the clinical characteristics which predict the response to antiarrhythmic drugs are not well described.

Methods: We analysed pts in the MUSTT study randomized to antiarrhythmic therapy. All pts had coronary disease, an ejection fraction (EF) <0.40, unsustained VT and inducible sustained VT or fibrillation (VF).

Results: There were 127 drug responders (38%) of the 330 pts tested. Of the responders, 88 (69%) were suppressed with the first randomized drug tested. The induction of VF at baseline was associated with a higher response rate than the induction of monomorphic VT (67% vs 34%, p = 0.001), and the median cycle length of monomorphic VT was shorter in responders than non-responders (240 vs 250 ms, p = 0.025). However, clinical characteristics such as age, gender, EF, heart failure (CHF), previous revascularization or extent of coronary disease did not predict drug response (p's > 0.05).

	Responders	Nonresponders
Age (yrs)	68	67
Gender (%male)	94	89
EF	0.30	0.30
CHF (%)	67	80

Conclusion: The inducible rhythm, but not clinical characteristics, correlate with antiarrhythmic drug response in this population.

1073-174 Electrophysiology Study Characteristics: Correlation With Cardiac Death and Defibrillator Shocks in the Multicenter Automated Defibrillator Implantation Trial

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Pts with CAD, poor LV function and unsustained VT are at a higher risk of death when sustained ventricular arrhythmia is inducible and non-suppressible at electrophysiology study (EPS). The association between the method of induction, the nature of the induced VT/VF & the outcome has not been studied. We used the MADIT database to compare survivors (n = 142) with cardiac deaths (n = 38), and pts with defibrillators who never received shocks (n = 40) to those who did (n = 62) over 1–61 mo. (mean 27)